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Differential Expression of Adipose Triglyceride Lipase and Long-Chain Acyl-CoA Synthetase 1 Genes in Geese and Their Regulation of Fat Deposition and Serum Lipid Metabolism: Postprint

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Abstract

This study aimed to investigate the differential expression of adipose triglyceride lipase (ATGL) and long-chain acyl-CoA synthetase 1 (ACSL1) genes in various tissues and organs of geese, and to explore their regulatory roles in body fat deposition and serum lipid metabolism. Thirty 16-week-old Wulong geese (half male and half female) were selected, and after slaughter, real-time quantitative PCR was used to detect the expression levels of ATGL and ACSL1 genes in different tissues and organs (liver, heart, subcutaneous fat, abdominal fat, breast muscle, leg muscle, muscular stomach, glandular stomach, small intestine, kidney, brain, lung, spleen). The results showed that: 1) Expression of both ATGL and ACSL1 genes was detected in subcutaneous fat, abdominal fat, liver, spleen, kidney, heart, breast muscle, and leg muscle of geese; ATGL gene expression was highest in subcutaneous fat and abdominal fat, followed by liver and spleen, with only low expression in kidney, heart, breast muscle, and leg muscle; ACSL1 gene expression was relatively high in subcutaneous fat, abdominal fat, liver, and spleen, with low expression in kidney, heart, breast muscle, and leg muscle, and almost no expression in muscular stomach, glandular stomach, and lung. 2) ATGL gene expression showed significant or highly significant negative correlations with leg muscle intramuscular fat percentage, breast muscle intramuscular fat percentage, abdominal fat percentage, breast muscle percentage, and leg muscle percentage ($P<0.05$ or $P<0.01$), and a significant positive correlation with subcutaneous fat percentage ($P<0.05$); ACSL1 gene expression showed positive correlations with leg muscle intramuscular fat percentage, breast muscle intramuscular fat percentage, and breast muscle percentage ($P>0.05$), a significant positive correlation with leg muscle percentage ($P<0.05$), and a significant negative correlation with subcutaneous

fat percentage (P<0.05). 3) ATGL gene expression showed significant or highly significant positive correlations with serum triglyceride, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and glucose contents (P<0.05 or P<0.01); ACSL1 gene expression showed negative correlations with serum total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and glucose contents (P>0.05), and a significant negative correlation with triglyceride content (P<0.05). These results indicate that ATGL and ACSL1 genes exhibit distinct differential expression patterns in different tissues and organs of geese, and exert opposite regulatory effects on body fat deposition and serum lipid metabolism.

Full Text

Differential Expression of Adipose Triglyceride Lipase and Long-Chain Acyl-CoA Synthetase 1 Genes and Their Regulation of Fat Deposition and Serum Lipid Metabolism in Geese

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Abstract: This study investigated the differential expression of adipose triglyceride lipase (ATGL) and long-chain acyl-CoA synthetase 1 (ACSL1) genes across various tissues and organs in geese, and explored their regulatory roles in fat deposition and serum lipid metabolism. Thirty 16-week-old Wulong geese (equal numbers of males and females) were selected and slaughtered. Real-time quantitative PCR was used to detect ATGL and ACSL1 gene expression in different tissues and organs, including liver, heart, subcutaneous fat, abdominal fat, breast muscle, leg muscle, muscular stomach, glandular stomach, small intestine, kidney, brain, lung, and spleen. The results revealed three key findings. First, ATGL and ACSL1 genes were expressed in subcutaneous fat, abdominal fat, liver, spleen, kidney, heart, breast muscle, and leg muscle. ATGL expression was highest in subcutaneous and abdominal fat, followed by liver and spleen, with only low levels detected in kidney, heart, breast muscle, and leg muscle. ACSL1 showed high expression in subcutaneous fat, abdominal fat, liver, and spleen, minimal expression in kidney, heart, breast muscle, and leg muscle, and was almost undetectable in muscular stomach, glandular stomach, and lung. Second, ATGL expression exhibited significant or highly significant negative correlations with leg muscle intramuscular fat percentage, breast muscle intramuscular fat percentage, abdominal fat percentage, breast muscle percentage, and leg mus-

cle percentage ($P<0.05$ or $P<0.01$), but a significant positive correlation with subcutaneous fat percentage ($P<0.05$). ACSL1 expression showed positive correlations with leg muscle intramuscular fat percentage, breast muscle intramuscular fat percentage, and breast muscle percentage ($P>0.05$), a significant positive correlation with leg muscle percentage ($P<0.05$), and a significant negative correlation with subcutaneous fat percentage ($P<0.05$). Third, ATGL expression was significantly or highly significantly positively correlated with serum triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and glucose content ($P<0.05$ or $P<0.01$). ACSL1 expression was negatively correlated with serum total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and glucose content ($P>0.05$), and significantly negatively correlated with triglyceride content ($P<0.05$). These findings demonstrate that ATGL and ACSL1 genes exhibit distinct differential expression patterns across various tissues and organs in geese, exerting opposing regulatory effects on fat deposition and serum lipid metabolism.

Keywords: geese; ATGL gene; ACSL1 gene; tissues and organs; gene expression; gene regulation

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Adipose triglyceride lipase (ATGL) is a recently discovered lipase that primarily catalyzes the first step of triglyceride hydrolysis in vivo [1], participating in the process of fat mobilization. It prevents excessive fat accumulation in the body and regulates growth and development [2]. Long-chain fatty acids serve as crucial metabolic substrates for energy acquisition and fat synthesis in organisms. Long-chain acyl-CoA synthetase 1 (ACSL1) utilizes long-chain fatty acids, ATP, and coenzyme A (CoA) as substrates to synthesize long-chain acyl-CoA esters, thereby regulating overall fatty acid metabolism through control of fatty acid esterification reactions. Zimmermann et al. [1] found that ATGL expression is highest in mouse adipose tissue, followed by testis, cardiac muscle, and skeletal muscle. Dai et al. [3] reported that ATGL expression is highest in pig backfat, followed by muscle, small intestine, and heart. In chickens [4] and ducks [5], ATGL expression is also highest in adipose tissue. Wang et al. [6] demonstrated that in Landes geese, ATGL expression in subcutaneous fat and brain is significantly higher than in other tissues. ACSL1 expression varies considerably across mammalian tissues. Suzuki et al. [7] first cloned ACSL1 in rat liver and detected its mRNA in heart and adipose tissue. Mashek et al. [8] found that ACSL1 is highly expressed in brown adipose tissue, gonads, liver, and heart, with low expression in skeletal muscle and brain. Lü [9] showed that in Sichuan white geese, ACSL1 expression is extremely high in abdominal and subcutaneous fat. Wulong geese (also known as Huoyan geese) are among the smallest goose breeds worldwide, characterized by high egg production, low abdominal fat content, and slower fat deposition rates compared to large goose breeds. Currently, few studies have investigated the expression of ACSL1 and ATGL genes in goose tissues and organs and their relationship with fat deposition and serum lipid metabolism. Therefore, this study used 5- to 16-week-old Wulong geese

as experimental subjects to investigate ATGL and ACSL1 gene expression in 13 tissues and organs including liver, spleen, abdominal fat, and subcutaneous fat under normal physiological conditions using fluorescence quantitative PCR. The objectives were to explore the differential expression patterns of ATGL and ACSL1 genes in different goose tissues and to preliminarily investigate the regulatory mechanisms of these functional genes in goose growth, development, and fat metabolism through correlation analysis with fat deposition, muscle growth, and fat metabolism.

1.1 Experimental Animals

Wulong geese were raised from 5 to 16 weeks of age under identical feeding conditions. At 16 weeks, 30 experimental geese (half male and half female) with non-significant differences in average body weight ($P>0.05$) were selected for slaughter and sampling.

1.2 Basal Diet

The basal diet was formulated according to NRC (1994) poultry nutrient requirements. Its composition and nutrient levels are shown in Table 1 . High-performance liquid chromatography determined the pantothenic acid content of the basal diet to be 9.45 mg/kg.

1.3 Feeding Management

Before the experiment, the goose house was thoroughly disinfected. Geese were raised in net-separated pens under indoor conditions throughout the experimental period. They had free access to water and feed, with frequent small additions, and their growth status was monitored regularly.

1.4.2 Determination of Fat Percentage in Leg Muscle, Breast Muscle, and Liver

Samples were removed from the -20°C freezer, thawed, minced, and weighed. They were dried in a 105°C oven and their absolute dry weight was measured using an electronic balance. Defatted filter paper was dried at 105°C and weighed. Samples were then ground, approximately 2 g was wrapped in filter paper, oven-dried to absolute dryness, and accurately weighed. The wrapped filter paper was placed in a Soxhlet extractor and extracted with ether for 12 hours at a water bath temperature of 70-80°C. After defatting, the sample packets were dried and weighed.

Sample (absolute dry) fat percentage (%) = 100 × (pre-defatting sample packet weight - post-defatting sample packet weight) / (pre-defatting sample packet weight - absolute dry filter paper weight) [10].

1.4.3 Serum Biochemical Indices

At the end of week 16, 5 mL of blood was collected from the wing vein and centrifuged at 3,000 r/min for 10 minutes to obtain serum. Serum triglyceride, total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol contents were measured using assay kits purchased from Nanjing Jiancheng Bioengineering Institute.

1.4.4 Quantitative Determination of Gene Expression

Liver samples were removed from liquid nitrogen, and 50-100 mg was quickly excised and mixed with 1 mL RNAisoTM Plus reagent for total RNA extraction according to the manufacturer' s instructions. Total RNA concentration and purity were checked using a spectrophotometer (UV-1100), and quality was assessed by agarose gel electrophoresis. RNA solutions were diluted to 1.0 g/ L.

RNA was immediately reverse-transcribed using the PrimeScriptTM RT reagent Kit (Takara, DRR037D) according to the kit instructions. The 20 L reverse transcription reaction contained: 10 L 2×RT Buffer, 1 L random primer (100 pmol/ L), 1 L RT-mix, 5 L template (RNA), and 3 L diethylpyrocarbonate (DEPC) water. Reaction conditions were: gentle mixing; 25°C for 10 min, 42°C for 60 min, and 85°C for 5 min. Reverse transcription products (cDNA) were stored at -20°C.

Primers were designed using Primer5.0 software based on goose ATGL mRNA sequence (GenBank accession No. HQ914789) and ACSL1 mRNA sequence (GenBank accession No. GQ891991) compared with the complete goose genome sequence. ATGL forward primer: 5 -TCGCAACCTCTACCGCCTCT-3 , reverse primer: 5 -TCCGCACAAGCCTCCATAAGA-3 . ACSL1 forward primer: 5 -GGAGGAAGAGTAAGGCTGATGGT-3 , reverse primer: 5 -CCAGGAACCGACAGTGAGCAT-3 . β-actin served as the internal reference gene with forward primer: 5 -GTTCTTGACTCTGGCGATGG-3 and reverse primer: 5 -TAAGGTTTCAGGACAGCGGA-3 . The predicted amplicon length was approximately 130 bp. Primers were synthesized by Shanghai Sangon Biotech.

Quantitative PCR was performed on reverse transcription products using a real-time PCR instrument (Bio-Rad CFX). The 50 L PCR reaction contained: 25 L 2×PCR Buffer, 1.2 L primer (25 pmol/ L), 0.3 L SYBR Green I, 1 L template (cDNA), and 22.5 L DEPC water. Reaction conditions were: 94°C for 4 min; 35 cycles of 94°C for 20 s, 60°C for 30 s, and 72°C for 30 s. Amplification curves were observed.

1.5 Data Processing and Statistical Analysis

Experimental raw data were processed using the 2^(-ΔΔCt) method. Data were then analyzed using one-way ANOVA in SPSS 17.0, with significance tested by

LSD method. Results are expressed as "mean ± standard deviation." Correlation analysis was also performed between gene expression levels and other indices.

2.1 PCR Amplification of ATGL and ACSL1 Genes

Using extracted total RNA as template, cDNA was obtained by RT-PCR, and ATGL, ACSL1, and β-actin genes were amplified (Figure 1 [Figure 1: see original paper]). Agarose gel electrophoresis of total RNA showed two clear rRNA bands at 28S and 18S, with 28S band intensity approximately twice that of 18S, indicating intact total RNA. 1: adipose triglyceride lipase ATGL; 2: long-chain acyl-CoA synthetase 1 ACSL1; 3: β-actin β-actin.

2.2 Differential Expression Analysis of ATGL and ACSL1 Genes in Various Tissues

Using β-actin as the internal reference gene, RT-PCR was used to detect ATGL and ACSL1 gene expression in goose liver, heart, subcutaneous fat, abdominal fat, breast muscle, leg muscle, muscular stomach, glandular stomach, small intestine, kidney, brain, lung, and spleen. Results showed that ATGL expression was high in subcutaneous fat, abdominal fat, liver, and spleen, low in kidney, heart, breast muscle, and leg muscle, and almost undetectable in muscular stomach, glandular stomach, and lung. ACSL1 showed similar patterns with high expression in subcutaneous fat, abdominal fat, liver, and spleen, low expression in kidney, heart, breast muscle, and leg muscle, and almost no expression in muscular stomach, glandular stomach, and lung (Figure 2 [Figure 2: see original paper]).

M. DL2000 DNA molecular weight marker; 1: liver; 2: heart; 3: subcutaneous fat; 4: abdominal fat; 5: breast muscle; 6: leg muscle; 7: muscular stomach; 8: glandular stomach; 9: small intestine; 10: kidney; 11: brain; 12: lung; 13: spleen.

As shown in Figure 3 [Figure 3: see original paper], the amplification curves of ATGL and ACSL1 genes showed normal trends, and the melting curves displayed single peaks without other miscellaneous peaks, indicating good primer specificity, absence of primer dimers and other non-specific amplification products, and a stable, reliable detection system.

As shown in Figure 4 [Figure 4: see original paper], the amplification curves of ATGL and ACSL1 genes had clear inflection points, flat baselines, and typical S-shaped curves.

2.3 Expression Levels of ATGL and ACSL1 Genes in Different Tissues

Table 2 shows that ATGL and ACSL1 genes were expressed in almost all tissues, with the highest expression in abdominal and subcutaneous fat, low expression in kidney, heart, breast muscle, and leg muscle, and almost no expression in glandular stomach, muscular stomach, and lung. Specifically, ATGL expression

in subcutaneous fat was 24.57, 36.86, and 73.71 times higher than in muscular stomach, glandular stomach, and lung, respectively ($P<0.01$). ATGL expression in abdominal fat was 17.57, 26.36, and 52.71 times higher than in muscular stomach, glandular stomach, and lung, respectively ($P<0.01$). ACSL1 expression in subcutaneous fat was 6.65, 6.00, and 11.71 times higher than in muscular stomach, glandular stomach, and lung, respectively ($P<0.01$). ACSL1 expression in abdominal fat was 18.16, 16.39, and 32.00 times higher than in muscular stomach, glandular stomach, and lung, respectively ($P<0.01$).

These results demonstrate differential expression of ATGL and ACSL1 genes across these tissues, with the highest expression in abdominal and subcutaneous fat, suggesting that both genes regulate fat deposition.

2.4 Relationship Between ATGL and ACSL1 Expression and Fat Deposition and Muscle Growth

Tables 3 and 4 show that ATGL expression was highly significantly negatively correlated with leg muscle intramuscular fat percentage and breast muscle intramuscular fat percentage ($P<0.01$), significantly negatively correlated with abdominal fat percentage, breast muscle percentage, and leg muscle percentage ($P<0.05$), and significantly positively correlated with subcutaneous fat percentage ($P<0.05$). ACSL1 expression was positively correlated with leg muscle intramuscular fat percentage, breast muscle intramuscular fat percentage, and breast muscle percentage ($P>0.05$), significantly positively correlated with leg muscle percentage ($P<0.05$), and significantly negatively correlated with subcutaneous fat percentage ($P<0.05$). Abdominal fat percentage was only 1.55% and negatively correlated with both ATGL and ACSL1 expression ($P>0.05$), confirming the low-fat breed characteristics of Wulong geese.

These results indicate that ATGL and ACSL1 genes exert opposing regulatory effects on fat deposition and muscle growth in geese, with ATGL having a more significant impact on tissue fat deposition than ACSL1.

2.5 Correlation Between ATGL and ACSL1 Expression and Serum Lipid Metabolism Indices

Tables 5 and 6 show that ATGL expression was positively correlated with serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and glucose content, with a highly significant positive correlation with triglyceride content ($P<0.01$). ACSL1 expression was negatively correlated with serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and glucose content, with a significant negative correlation with triglyceride content ($P<0.05$).

These results demonstrate that ATGL and ACSL1 expression is closely associated with lipid metabolism, particularly triglyceride metabolism, with opposing regulatory effects.

3.1 Tissue Expression Specificity of ATGL and ACSL1 Genes

The ATGL protein contains a GXSXG motif with lipase activity and a special Patatin domain at its N-terminus that is highly conserved across species. ATGL shows highest expression in mouse adipose tissue, followed by testis, cardiac muscle, and skeletal muscle, with low-level expression in liver, spleen, kidney, brain, and lung [1]. Nie et al. [11-12] reported high ATGL expression in avian adipose tissue. Wang [6] found that ATGL expression in Landes geese was higher in subcutaneous fat and brain than in other tissues, with lowest expression in stomach and lung. Mashek et al. [8] reported that ACSL1 is highly expressed in brown adipose tissue, gonads, liver, and heart, with low expression in skeletal muscle and brain. Lü [9] showed that ACSL1 expression in Sichuan white geese was extremely high in abdominal and subcutaneous fat, moderate in liver and heart, and very low in brain.

This study detected ATGL expression in subcutaneous fat, abdominal fat, liver, spleen, kidney, heart, breast muscle, and leg muscle of geese, indicating broad expression across various tissues under normal physiological conditions, with highest expression in adipose tissue, consistent with Nie et al. [11]. ATGL expression was highest in subcutaneous and abdominal fat, similar to mammals and chickens/ducks [12], followed by liver and spleen, with low expression in kidney, heart, breast muscle, and leg muscle, and almost no expression in stomach, lung, and small intestine. This differential expression pattern suggests important biological significance of ATGL in adipose tissue. However, ATGL expression in spleen was at an intermediate level in this study, approximately 1/5 of that in abdominal fat, whereas Wang [6] reported spleen expression twice that of abdominal fat. Whether this discrepancy results from individual or breed differences requires further investigation.

This study also detected ACSL1 expression in subcutaneous fat, abdominal fat, liver, spleen, kidney, heart, breast muscle, and leg muscle of geese, consistent with Mashek et al. [8]. High expression in subcutaneous fat, abdominal fat, liver, and spleen, low expression in kidney, heart, breast muscle, and leg muscle, and almost no expression in muscular stomach, glandular stomach, and lung showed a pattern similar to Lü [9], with minimal sequence variation in functional regions, indicating functional conservation and a close relationship between ACSL1 and fatty acid synthesis/metabolism. Overexpression of ACSL1 in human HepG2 cells [13] and rat liver [14] and heart [15] promotes fatty acid conversion to triglycerides. Immunoprecipitation studies have shown interaction between fatty acid transport protein 1 (FATP1) and ACSL1 genes, promoting cellular fatty acid uptake [16]. The expression pattern observed in this study suggests that ACSL1 functions to promote triglyceride deposition and fatty acid transport.

3.2 Correlation Between ATGL and ACSL1 Expression and Body Fat Deposition Indices

Slaughter performance indices reflect differences in nutrient deposition among tissues and within the same tissue. Zimmermann et al. [1] found that ATGL expression is closely related to adipose tissue development. Muoio et al. [17] reported that long-chain acyl-CoA is an essential substrate for lipid and protein synthesis, and ACSL1 inhibition reduces triglyceride content in mice. Kim et al. [4] showed that peroxisome proliferator-activated receptor (PPAR) agonists in liver can induce ACSL1 expression and increase related enzyme activities. This study found negative correlations between ATGL and ACSL1 expression and abdominal fat percentage, consistent with Zimmermann et al. [1], confirming that ACSL1 and ATGL play active roles in long-chain fatty acid synthesis and body development.

Cui et al. [18] reported that the liver is an important organ for fat synthesis, with significantly increased activity of fat metabolism-related enzymes during growth and development. The ACSL1 promoter region contains PPAR response elements [19] that mediate signal transduction pathways involved in protein synthesis to regulate growth. ATGL regulates insulin secretion and sensitivity through modulation of interleukins, transcription factor-binding proteins, and uncoupling protein 1 in fat metabolism pathways [20], promoting fat lipolysis, reducing adipocyte number, and slowing intramuscular fat increase relative to muscle growth. This study showed that ATGL expression was highly significantly negatively correlated with breast and leg muscle intramuscular fat percentages, while ACSL1 expression was positively correlated with these parameters, indicating opposing regulation of intramuscular fat percentage, with ATGL having a more significant regulatory effect. Intramuscular fat consists mainly of phospholipids and triglycerides, and ATGL, as the rate-limiting enzyme for triglyceride hydrolysis, helps prevent excessive fat deposition in breast and leg muscle, reducing obesity and related disorders. Wulong geese selected for this study showed low abdominal fat content and slow fat deposition, possibly due to ATGL and ACSL1 regulation of fat metabolism-related enzyme activity in liver affecting adipocyte development in muscle, though the mechanism requires further study.

Yin et al. [21] suggested that higher intramuscular fat content in goose leg muscle compared to breast muscle is related to adipocyte growth and development, ACSL1 activity, and microvascular tissue development at different sites. This study confirmed that Wulong geese also have higher leg muscle intramuscular fat percentage than breast muscle, with ACSL1 expression detected in both tissues, indicating its important regulatory role in intramuscular fat metabolism.

3.3 Correlation Between ATGL and ACSL1 Expression and Serum Lipid Metabolism Indices

Poultry fat development and deposition are primarily affected by serum triglyceride content [22]. Wen et al. [23] found in Beijing ducks that when secretion rates of high-density and very low-density lipoproteins are lower than triglyceride synthesis rates, fat accumulates extensively in the liver and serum triglyceride content increases significantly. Zechner et al. [25] confirmed that ATGL catalyzes the first and rate-limiting step of the entire lipolysis process. In mammals, ACSL1 is mainly expressed in adipose tissue and liver, localized to the inner cell membrane, and involved in triglyceride synthesis and fatty acid uptake [26]. ATGL participates in the first step of triglyceride hydrolysis, generating diglycerides and free fatty acids [27]. ACSL1 uses long-chain fatty acids (C12-C22), ATP, and CoA as substrates to generate corresponding long-chain acyl-CoA esters [28], which are important substrates for triglyceride synthesis. This study showed that ATGL expression was highly significantly positively correlated with triglyceride content, while ACSL1 expression was significantly negatively correlated, indicating that both genes play active roles in promoting lipid synthesis and fatty acid transport in Wulong geese fat metabolism, with opposing regulatory effects.

Glucose is the primary method for energy accumulation and conversion in organisms. Li [24] reported in broiler breeder offspring that glucose is the main functional substance and precursor for fat synthesis in animals, with serum glucose content affected by the degree of fat metabolism. The liver is the hub for glucose metabolism regulation [29]. ATGL and ACSL1 genes participate in hepatic glucose and fat metabolism, regulating triglyceride and related protein synthesis and decomposition [30], releasing glycerol and free fatty acids, participating in the tricarboxylic acid cycle to affect glucose content, and can effectively reduce fat generation and accumulation and promote energy consumption by inhibiting insulin secretion, maintaining normal physiological functions. This study showed that ATGL expression was positively correlated with glucose content, while ACSL1 expression was negatively correlated, indicating opposing regulation of glucose metabolism by these two genes. However, Wang et al. [31] found no significant correlation between glucose content and lipid metabolism genes in broilers, suggesting that whether this discrepancy is species-related or due to experimental error requires further investigation.

Conclusion

This study demonstrates four key findings. First, ATGL and ACSL1 genes exhibit distinct differential expression patterns across various tissues and organs in Wulong geese, with highest expression in abdominal and subcutaneous fat, indicating regulatory roles in fat deposition. Second, ATGL expression is negatively correlated with leg muscle intramuscular fat percentage, breast muscle intramuscular fat percentage, breast muscle percentage, and leg muscle percentage, but significantly positively correlated with subcutaneous fat percentage. Conversely,

ACSL1 expression is positively correlated with leg muscle intramuscular fat percentage, breast muscle intramuscular fat percentage, breast muscle percentage, and leg muscle percentage, but significantly negatively correlated with subcutaneous fat percentage. Third, ATGL expression is positively correlated with serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and glucose content, while ACSL1 expression is negatively correlated with these parameters, particularly showing a strong relationship with triglyceride metabolism. Fourth, ATGL and ACSL1 gene expression exerts opposing regulatory effects on fat deposition and serum lipid metabolism in geese.

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